IN THE SPECIFICATION:

Please amend paragraph [0061] as follows:

[0061] Optionally, one or more compounds having adjuvant activity may be added to the vaccine. Suitable adjuvants are for example aluminium hydroxide, phosphate or oxide, oilemulsions (e.g. of Bayol F® or Marcol 52® BAYOL F® or MARCOL 52®), saponins or vitamin-E solubilisate.

Please amend paragraphs [0100] through [0104] as follows:

[0100] FIG. 1. Target cell sensitization of alanine replacement epitopes. (A) Chromium labeled T2 target cells were preincubated for 1 hour with various amounts of the indicated alanine-substituted epitope-analogues (AAGIGILTV (SEQ ID NO:8), GAGIGILTV (SEQ ID NO:10), and AAGIGILTA (SEQ ID NO:25)). Melan-A/MART-1 27-35-reactive TIL 1235 lymphocytes were added at an effector to target ratio of 20. (B) Target cell sensitization of alanine-substituted gp100 154-162-analogues was analyzed using gp100-reactive TIL 1200 lymphocytes at an effector to target ratio of 20 (KTWGQYWQV (SEQ ID NO:9), ATWGQYWQV (SEQ ID NO:11), KAWGQYWQV (SEQ ID NO:13), KTAGQYWQV (SEQ ID NO:15), KTWAQYWQV (SEQ ID NO:17), KTWGQYWQV (SEQ ID NO:1), and KTWGQYWQA (SEQ ID NO:26)).

[0101] FIG. 2. Target cell sensitization of N-terminal anchor-replacement epitopes. Chromium release experiments were performed as in FIG. 1. (A) Melan-A/MART-1 27-35-reactive TIL 1235 lymphocytes were used to assay target cell sensitization by the Melan-A/MART-1 27-35 analogues (AAGIGILTV (SEQ ID NO:8), AVGIGILTV (SEQ ID NO:27), ALGIGILTV (SEQ ID NO:28), and AIGIGILTV (SEQ ID NO:30)). (B) Gp100 154-162-reactive TIL 1200 lymphocytes were used to assay target cell sensitization by the gp100 154-162-analogues (KTWGQYWQV (SEQ ID NO:9), KVWGQYWQV (SEQ ID NO:2), KLWGQYWQV (SEQ ID NO:3), KMWGQYWQV (SEQ ID NO:35), and KIWGQYWQV (SEQ ID NO:4)).

[0102] FIG. 3. Immunogenicity of gp100 154-162 epitope-analogues in HLA-A*0201/K^b transgenic mice (KTWGQYWQV (SEQ ID NO:9), KTWGQYWAV (SEQ ID NO: 1), KVWGQYWQV (SEQ ID NO:2), KLWGQYWQV (SEQ ID NO: 3), and KIWGQYWQV (SEQ ID NO: 4)). Bulk CTL obtained from immunized mice were tested for lytic activity using chromium labeled Jurkat A2/K^b target cells that were preincubated with no peptide, 10mM wild type gp100 154-162 or 10mM of the epitope-analogue used to immunize the mice. For each peptide the mean specific lysis of bulk CTL of the responding mice is shown. Standard deviations never exceeded 15% of the mean value. One representative experiment out of two is shown.

[0103] FIG. 4. Peptide specific reactivity of *in vitro* induced epitope-analogue specific CTL cultures (KTWGQYWQV (SEQ ID NO:9), KTWGQYWAV (SEQ ID NO:1), KVWGQYWQV (SEQ ID NO:2), KLWGQYWQV (SEQ ID NO:3), and KIWGQYWQV (SEQ ID NO:4)). Chromium-labeled HLA-A*0201⁺ T2 target cells were pre-incubated with 10 mM of an irrelevant HLA-A*0201-binding peptide, 10mM wild type gp100 154-162 or 10mM of the epitope-analogue used for CTL induction. The different CTL cultures were added at an effector to target ratio of 20:1. One representative experiment out of two is shown.

[0104] FIG. 5. Epitope-analogue induced CTL cultures specifically lyse melanoma cells endogenously presenting the wild type epitope (KTWGQYWQV (SEQ ID NO:9), KTWGQYWAV (SEQ ID NO:1), KVWGQYWQV (SEQ ID NO:2), KLWGQYWQV (SEQ ID NO:3), and KIWGQYWQV (SEQ ID NO:4)). Chromium-labeled HLA-A2.1⁺ BLM and Mel 624 melanoma cells were used as target cells. BLM cells lack expression of gp100. The different CTL cultures were added at an effector to target ratio of 20:1. One representative experiment out of two is shown.

Please amend paragraph [0106] as follows:

[0106] Healthy caucasian volunteers were phenotyped HLA-A2 by flow cytometry using mAbs BB7.2 (Parham et al., 1981, *Hum. Immunol. 3:277*) and MA2.1 (Parham et al., 1978, *Nature 276:397*). The donors underwent leukapheresis and PBMC were isolated by Ficoll/Hypaque FICOLLTM (branched hydrophilic polysaccharide) /HYPAQUE® (diatrizoic acid) density gradient centrifugation. The cells were cryopreserved in aliquots of 4 x 10⁷ PBMC.

Please amend paragraph [0110] as follows:

[0110] Peptide-binding to HLA-A*0201 was analyzed using HLA-A*0201⁺ JY cells as was described previously (van der Burg et al., 1995, *Hum. Immunol. 44:189*). Briefly, mild-acid treated JY cells were incubated with 150 nM Fluorescein (FL)-labeled reference peptide (FLPSDC(-FL)FPSV) (SEQ ID NO:6) and with several concentrations of competitor peptide for 24 hours at 4° in the presence of 1.0 mg/ml β2-microglobulin (Sigma, St. Louis, MO). Subsequently, the cells were washed, fixed with paraformaldehyde and analyzed by flow cytometry. The mean-fluorescence (MF) obtained in the absence of competitor peptide was regarded as maximal binding and equated to 0%; the MF obtained without reference peptide was equated to 100% inhibition. % inhibition of binding was calculated using the formula: (1-(MF 150 nM reference & competitor peptide - MF no reference peptide) ÷ (MF 150 nM reference peptide- MF no reference peptide) x 100%. The binding capacity of competitor peptides is expressed as the concentration needed to inhibit 50% of binding of the FL-labeled reference peptide (IC₅₀).

Please amend paragraph [0112] as follows:

[0112] Groups of 3 HLA-A*0201/K^b transgenic mice were injected subcutaneously in the base of the tail vein with 100 mg peptide emulsified in IFA in the presence of 140 mg of the H-2 I-A^b-restricted HBV core antigen-derived T helper epitope (128-140; sequence TPPAYRPPNAPIL (SEQ ID NO:5)) (Milich et al., 1988, *Proc. Natl. Acad. Sci. U. S. A.* 85:1610). After 11 days, mice were sacrificed and spleen cells (30 x 10⁶ cells in 10 ml) were restimulated in vitro with peptide-loaded syngeneic irradiated LPS-stimulated B cell lymphoblasts (ratio 4:1). At day 6 of culture, the bulk responder populations were tested for specific lytic activity.